A recent report from the UK-TIA Study Group\(^1\) presented 11 cases of intracranial tumours among 2449 patients with transient ischaemic attacks or minor strokes. Occasional cases of small cerebral haematomas have been found in patients with minor strokes,\(^2\) but only exceptionally in patients with transient ischaemic attacks.\(^3\) In a CT scan study of 284 cases with transient ischaemic attacks\(^4\) five patients had a mass lesion; none a brain haematoma.

We present the results of a prospective CT scan study of 175 patients (63 with transient ischaemic attacks and 112 with minor deficits lasting longer than 24 hours) recruited in the emergency rooms of two general hospitals. In every case, the CT scan (CX Tomoscan, Philips) was performed within the first week of the clinical event (with a mean delay of 13 (SD 24) hours). The mean age of the patients was 68 (8.5) years; 132 events were located in the carotid artery territory, 38 in the verteobasilar territory and five were of uncertain location. The CT scan was normal in 114 patients. Low density areas compatible with infarction were present in 56. Non-ischaemic causes of the presenting symptoms were found in three minor cases of stroke—namely; a brain tumour, compatible with an extensive hemispheric malignant glioma, with biopsy features; a small occipital haematoma; and a medium sized basal ganglia haematoma (associated with an ipsilateral subdural haematoma). All three patients were over 60 and had at least one vascular risk factor. In one of the cases of transient ischaemic attack a mass located on the clivus (compatible by CT features with a meningioma) was considered to have caused cerebral symptoms through compression of the basilar artery. Also, aetiologically related to the symptoms could have been a minor stroke case with a thombosed middle cerebral artery bifurcation aneurysm, demonstrated by MRI angiography.

The number of cases in our study is insufficient to support definite conclusions. The yield of CT scan for the detection of non-ischaemic causes (such as cerebral and subdural haematoma and brain tumour) in minor strokes (cases with symptoms lasting longer than 24 hours) was 2.7% (95% CI 0.0 to 5.7) and in transient ischaemic attacks the yield was 1.6% (95% CI 0.0 to 4.7).

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Sumatriptan and giant cell arteritis

We have proposed a unified theory that suggests that migraine is essentially driven from the central nervous system and entrains the trigeminal innervation of the cranial vessels to form one of the clinical expressions of the disease.\(^5\) The recent into clinical practice of the novel antimigraine compound sumatriptan, a serotonin (5-HT) agonist, has provided a tool to understand further the underlying mechanisms of the disease. Its action as a vasoconstrictor and its inhibition of neurogenic inflammation in experimental animals has been cited by various groups as evidence for either the vascular or neurogenic inflammatory theories of migraine respectively. A patient was recently admitted to our institution with giant cell arteritis and headache not responsive to sumatriptan. Her lack of response casts some doubt on the neurogenic inflammatory theory of migraine.

The patient is a 68 year old woman who had a 10 day history of right sided temporal and frontal headache. The headache had spread from a small region above the eye to involve most of the right side of the head and she had noticed some increasing tenderness of the scalp muscles. The headache became more severe for 7 days and had some pounding exacerbations but no associated features of migraine. Eight days into the illness she attended her general practitioner and was given sumatriptan (100 mg) orally as a single dose, which did not alter the headache. She had no other history, particularly of regular headaches, and there was no relevant family history. Physical examination was unremarkable except for tenderness of the temporal arteries bilateral-ly. The erythrocyte sedimentation rate (ESR) at this time was 110 mm/h. She was treated with high dose steroids with complete remission of her headache and general malaise and a drop in the ESR by the next day. A temporal artery biopsy showed pronounced inflammatory changes.

The patient had a personal presentation of temporal arteritis that responded to steroids and she had remained well on steroids. She had no response to sumatriptan despite some side effects from the drug, notably nausea and mild neck and arm discomfort typical of that reported in trials. Practitioners should be watchful for secondary headache and in the elderly tempo-ral arteritis should be considered. Administration of a cranial vasoconstrictor to patients with inflamed narrowed vessels with the propensity to thrombose must be avoided absolutely. Sumatriptan is not a bedside test for migraine; it must not replace the careful history and should only be given to patients with a positive diagnosis in appropriate circumstances.

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Isolated lingual myoclonus associated with an Arnold-Chiari malformation

Reports on isolated rhythmic tongue movements are infrequent and may contribute to the understanding of rhythmic hyperkinet-ica in general. Isolated rhythmic move-ments of the tongue led to the diagnosis of an Arnold-Chiari malformation.

Case report

A 61-year-old man noted continuous jerking movements of the tongue for the first time 15 days before he was admitted to Sant Pau Hospital in 1984. The jerks, which were not preceded by any illness, persisted all day, were not accompanied by a grunting noise, and had not stopped or influenced by any action attempted by the patient. In 1974 he had a reactive mental depression that was treated with amitriptyline (75 mg/day) for nine months. No other medication was taken regularly in the nine years before the beginning of the abnormal tongue movements. There were no antecedents of head trauma or other rele-vant personal or family history. Neuro-logical examination was normal apart from the lingual jerks. They consisted of continu-ous rhythmic 3 Hz, low amplitude, symmet-rical contractions of both lateral edges of the tongue affecting the anterior and posterior parts and causing a midline depression of the tongue. The soft palate and other muscles innervated by the brain-stem were not involved. The jerks persisted during sleep. Although he did not complain about phonation disturbances, speech was mildly affected. A forced palatalisation of some words was evident. He spontaneously produced vowel prolongations and the tongue pulled against the palate; when he was asked to let the tongue free in the mouth, most sounds had a quavering quality. Sustained phonation, intrusion, passive depression, touching the tips to the tongue did not influence the amplitu-de or the rhythm of the jerks. After hours of continuous activity, the movements would unexpectedly stop, only to start once again after a few minutes.

Routine blood analysis, surface ECG, cortical somatosensory evoked potentials, and brain stem auditory evoked responses were all normal. A CT scan showed a dis-fuse and symmetrical enlargement of the two lateral and third ventricles with mild periventricular oedema and a normal sized fourth ventricle. An Arnold-Chiari type I malformation was evident on an MRI study (figure).\(^6\) After three days on 6 mg/day of clon-azeepam the movements were no longer con-tinuous but occurred, at the same rate and amplitude, in random bursts lasting up to three minutes. These bursts totally dis-appeared in the second week. Over the next eight years, many attempts to discontinue clonazeepam were followed by the re-emer-gence of the movements. During this period, other neurological and neuropsy-chological examinations, as well as repeated EEGs during and between the episodes,